IN THE UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Appl. No.	:	10/629,975	Confirmation No. 9513
Applicant	:	James Hunter Boone	
Filed	:	07/30/2003	
Title	:	METHOD FOR DIFFERENTIATING IRRITABLE BOWEL SYNDROME FROM INFLAMMATORY BOWEL DISEASE (IBD) AND FOR MONITORING PERSONS WITH IBD USING TOTAL ENDOGENOUS LACTOFERRIN AS A MARKER	
Group Art Unit	:	1641	
Examiner	:	Lisa V. Cook	
Docket No.	:	TLAB.109338	
Customer No.	:	05251	

VIA EFS-WEB SUBMISSION – March 12, 2009

Mail Stop Appeal Brief-Patents
Commissioner for Patents
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APPELLANTS' APPEAL BRIEF

This is an appeal from an Office Action dated September 12, 2008, rejecting claims 1, 2, and 6. These claims have been at least twice rejected. Appellants filed a Notice of Appeal and a Pre-Appeal Brief on December 12, 2008 within the time period provided under § 1.134 accompanied by the fee set forth in 37 C.F.R. § 41.20(b)(1). It is hereby requested that the time period for filing the Appeal Brief be extended for one month, or until March 12, 2009. Submitted herewith is the Appeal Brief, along with the fee set forth in §41.20(b)(2). The Commissioner is hereby authorized to charge any additional fee that may be due, or credit any overpayment, to Deposit Account No. 19-2112, referencing attorney docket number TLAB.109338.

Contents

I. REAL PARTY IN INTEREST

The real party in interest is TechLab, Inc., a corporation of the State of Virginia, United States of America.

II. RELATED APPEALS AND INTERFERENCES

None.

III. STATUS OF CLAIMS

Claims 1, 2, and 6 are pending and rejected, and the rejection of each of claims 1, 2, and 6 is being appealed. Originally filed claims 3-5 have been canceled.

IV. STATUS OF AMENDMENTS

No amendments have been filed subsequent to the Office Action dated September 12, 2008.

V. SUMMARY OF CLAIMED SUBJECT MATTER

The instant Application includes two independent claims: 1 and 6. The present invention is defined by the claims, but summarily, embodiments of the invention are generally directed to the clinical differentiation and monitoring of gastrointestinal illnesses. *See e.g., Specification*[1], p. 1, lines 12-13 (¶ [0003]). As described in the Specification of the present application, embodiments of the invention allow for monitoring a person having inflammatory bowel disease for gastrointestinal inflammation. *See id.* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. A first human fecal sample is collected from the person, diluted, and analyzed to determine the lactoferrin concentration. *See id.* at p. 32, lines 4-6 (¶ [0060]). Subsequently, a second human fecal sample is collected from the person, diluted, and analyzed to determine the lactoferrin concentration. *See id.* Lactoferrin concentration of the first sample is compared to the lactoferrin concentration of the second sample to monitor the inflammatory bowel disease activity of the person. *See id.*

[1] Please note that all references to the Specification refer to the Specification of the present application as filed on July 30, 2003.

Claim 1 (first of two independent claims)

Claim 1 is directed to a method for monitoring a person having inflammatory bowel disease for gastrointestinal inflammation. *See e.g., Specification*, p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method of claim 1 includes obtaining a first human fecal sample from a person; diluting the first fecal sample; contacting the first sample with immobilized polyclonal antibodies to endogenous lactoferrin to create a first treated sample; contacting the first treated sample with enzyme-linked polyclonal antibodies to create a first enzyme-linked antibody bound sample; adding a substrate to the first enzyme-linked antibody bound sample to create a first readable sample and determining the density of the first readable sample at 450nm; and generating a purified lactoferrin standard curve and determining a linear portion of the standard curve. *See id.* at p. 21, lines 1-23 to p. 22, lines 1-17 (¶¶ [0040]-[0044]); p. 31, lines 4-23 to p. 32, lines 1-2 (¶¶ [0058]-[0059]); p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method further includes comparing the optical density of the first readable sample to the standard curve to determine a concentration of the first diluted sample; and determining whether the concentration of the first diluted sample is within the linear portion of the standard curve. *See id.* at p. 31, lines 15-32 to p. 32, lines 1-2 (¶ [0059]). The method of claim 1 further includes determining the concentration of total endogenous lactoferrin in the first fecal sample. *See id.* Additionally, the method includes obtaining a second human fecal sample from the person at a time after the first sample was obtained. *See id.* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method of claim 1 further includes diluting the second fecal sample; contacting the second sample with immobilized polyclonal antibodies to endogenous lactoferrin to create a second treated sample; contacting the second treated sample with enzyme-linked polyclonal antibodies to create a second enzyme-linked antibody bound sample; adding a substrate to the second enzyme-linked antibody bound sample to create a second readable sample; determining the

optical density of the second readable sample at 450nm; comparing the optical density of the second readable sample to the standard curve to determine a concentration of the second diluted sample; and determining whether the concentration of the second diluted sample is within the linear portion of the standard curve. *See id.* at p. 31, lines 15-32 to p. 32, lines 1-2 (¶ [0059]); p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method further includes determining the concentration of total endogenous lactoferrin in the second fecal sample. *See id.* at p. 31, lines 15-32 to p. 32, lines 1-2 (¶ [0059]). The method of claim 1 further includes comparing the lactoferrin concentration of the first fecal sample to the lactoferrin concentration of the second sample for the person to monitor the inflammatory bowel disease activity of the person and to determine if the person has had a decrease or increase in gastrointestinal inflammation. *See id.* at p. 35, lines 10-18 (¶ [0062]).

Claim 6 (second of two independent claims)

Claim 6 is directed to a method for monitoring a human having inflammatory bowel disease for gastrointestinal inflammation. *See e.g., Specification* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method of claim 6 includes obtaining a first fecal sample from a human having inflammatory bowel disease at a first time. *See id.* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV; p. 35, lines 10-18 (¶ [0062]). Additionally, the method includes determining the concentration of endogenous lactoferrin in said first fecal sample to obtain a first lactoferrin concentration. *See id.* The method of claim 6 further includes obtaining a second fecal sample from the human having inflammatory bowel disease at a second time after the treatment of the human's inflammatory bowel disease later than said first time. *See id.* The method also includes determining the concentration of endogenous lactoferrin in said second sample to obtain a second lactoferrin concentration. *See id.* The method of claim 6 additionally includes comparing said first lactoferrin concentration to determine whether treatment of the

inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation. *See id.*

VI. GROUNDS OF REJECTIONS TO BE REVIEWED ON APPEAL

A) Whether claims 1, 2, and 6 are unpatentable over U.S. Patent No. 6,358,939 to Hayes et al. (the "Hayes reference") in view of Sreekant Murthy, PhD (Inflammation Research Association, Newsletter, September & December 1999, Vol. 9, No. 3 & 4, pages 1-14) (the "Murthy reference") in further view of U.S. Patent No. 5,552,292 to Uchida et al. (the "Uchida reference") and Aguila La O et al. (Biotecnologia Aplicada, Julio-Septembre, 2000, Vol. 17, No. 3, pages 177-182, English Abstract) (the "Aguila La O reference") under 35 U.S.C. § 103(a) .

B) Whether claims 1, 2, and 6 are unpatentable over U.S. Patent No. 6,358,939 to Hayes et al. (the "Hayes reference") in view of Sreekant Murthy, PhD (Inflammation Research Association, Newsletter, September & December 1999, Vol. 9, No. 3 & 4, pages 1-14) (the "Murthy reference") in further view of Sugi et al. (The American Journal of Gastroenterology, Vol. 91, No. 5, 927-934) (the "Sugi reference") and Aguila La O et al. (Biotecnologia Aplicada, Julio-Septembre, 2000, Vol. 17, No. 3, pages 177-182, English Abstract) (the "Aguila La O reference") under 35 U.S.C. § 103(a).

Appellants respectfully traverse the rejection of these claims.

VII. ARGUMENT

A) The rejection of claims 1, 2, and 6 under 35 U.S.C. § 103(a) as being unpatentable over the Hayes reference in view of the Murthy reference and in further view of the Uchida and Aguila La O references should be reversed because the Examiner has failed to establish a *prima facie* case of obviousness.

Initially, Applicants note that a patent shall not issue when "the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains." The Supreme Court in Graham v. John Deere

counseled that an obviousness determination is made by identifying: the scope and content of the prior art; the level of ordinary skill in the prior art; the differences between the claimed invention and prior art references; and secondary considerations.[2] To support a finding of obviousness, the initial burden is on the Office to apply the framework outlined in Graham and to provide some articulated reason, suggestion, or motivation, found either in the prior art references themselves or in the knowledge generally available to one of ordinary skill in the art, to modify the prior art reference or to combine prior art reference teachings to produce the claimed invention.[3] The Supreme Court elaborated, at pages 13-14 of the *KSR* opinion, that "it will be necessary for [the Office] to look at interrelated teachings of multiple [prior art references]; the effects of demands known to the design community or present in the marketplace; and the background knowledge possessed by [one of] ordinary skill in the art, all in order to determine whether there was an apparent reason to combine the known elements in the fashion claimed by the [patent application]."[4] Accordingly, in order to establish a *prima facie* case of obviousness, the Office shall provide a "clear articulation of the reason(s) why the claimed invention would have been obvious" based on factual findings upon applying the *Graham* factual inquiries.[5]

Applicants respectfully submit that claims 1, 2, and 6 are patentable over the Hayes, Murthy, Uchida, and Aguila La O references. In particular, a *prima facie* case of obviousness has not been established for claims 1, 2, and 6 because the Hayes, Murthy, Uchida, and Aguila La O references, either alone or in combination, fail to teach or suggest all the claim limitations for claims 1, 2, and 6. As such, the claim rejections are improper and should be withdrawn.

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[2] *Graham v. John Deere Co.*, 383 U.S. 1 (1966).
[3] *See, Application of Bergel*, 292 F. 2d 955, 956-957 (1961).
[4] *KSR v. Teleflex*, No. 04-1350, 127 S.Ct. 1727 (2007).
[5] MPEP § 2143

Independent Claims 1 and 6

As noted above, independent claim 1 is directed to a method for monitoring a person having inflammatory bowel disease for gastrointestinal inflammation. *See, e.g., Specification*, p. 32, lines 4-6 (¶ 0060); p. 32, Table XV. The method of claim 1 includes obtaining a first human fecal sample from a person; diluting the first fecal sample; contacting the first sample with immobilized polyclonal antibodies to endogenous lactoferrin to create a first treated sample; contacting the first treated sample with enzyme-linked polyclonal antibodies to create a first enzyme-linked antibody bound sample; adding a substrate to the first enzyme-inked antibody bound sample to create a first readable sample and determining the density of the first readable sample at 450nm; generating a purified lactoferrin standard curve and determining a linear portion of the standard curve. *See id.* at p. 21, lines 1-23 to p. 22, lines 1-17 (¶¶ [0040]-[0044]); p. 31, lines 4-23 to p. 32, lines 1-2 (¶¶ [0058]-[0059]); p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method further includes comparing the optical density of the first readable sample to the standard curve to determine a concentration of the first diluted sample; and determining whether the concentration of the first diluted sample is within the linear portion of the standard curve. *See id.* at p. 31, lines 15-32 to p. 32, lines 1-2 (¶ [0059]). The method of claim 1 further includes determining the concentration of total endogenous lactoferrin in the first fecal sample. *See id.* Additionally, the method includes obtaining a second human fecal sample from the person at a time after the first sample was obtained. *See id.* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method of claim 1 further includes diluting the second fecal sample; contacting the second sample with immobilized polyclonal antibodies to endogenous lactoferrin to create a second treated sample; contacting the second treated sample with enzyme-linked polyclonal antibodies to create a second enzyme-linked antibody bound sample; adding a substrate to the second enzyme-linked antibody bound sample to create a second readable sample; determining

the optical density of the second readable sample at 450nm; comparing the optical density of the second readable sample to the standard curve to determine a concentration of the second diluted sample; and determining whether the concentration of the second diluted sample is within the linear portion of the standard curve. *See id.* at p. 31, lines 15-32 to p. 32, lines 1-2 (¶ [0059]); p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method further includes determining the concentration of total endogenous lactoferrin in the second fecal sample. *See id.* at p. 31, lines 15-32 to p. 32, lines 1-2 (¶ [0059]). The method of claim 1 further includes comparing the lactoferrin concentration of the first fecal sample to the lactoferrin concentration of the second sample for the person to monitor the inflammatory bowel disease activity of the person and to determine if the person has had a decrease or increase in gastrointestinal inflammation. *See id.* at p. 35, lines 10-18 (¶ [0062]).

Also noted above, claim 6 is directed to a method for monitoring a human having inflammatory bowel disease for gastrointestinal inflammation. *See e.g., Specification* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV. The method of claim 6 includes obtaining a first fecal sample from a human having inflammatory bowel disease at a first time. *See id.* at p. 32, lines 4-6 (¶ [0060]); p. 32, Table XV; p. 35, lines 10-18 (¶ [0062]). Additionally, the method includes determining the concentration of endogenous lactoferrin in said first fecal sample to obtain a first lactoferrin concentration. *See id.* The method of claim 6 further includes obtaining a second fecal sample from the human having inflammatory bowel disease at a second time after the treatment of the human's inflammatory bowel disease later than said first time. *See id.* The method also includes determining the concentration of endogenous lactoferrin in said second sample to obtain a second lactoferrin concentration. *See id.* The method of claim 6 additionally includes comparing said first lactoferrin concentration to determine whether treatment of the

inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation. *See id.*

Each of independent claims 1 and 6 recite, in part, limitations directed to obtaining a first fecal sample from a person and obtaining a second fecal sample from the same person, at a time subsequent to obtaining the first fecal sample. Independent claims 1 and 6 further recite comparing the lactoferrin concentration of the first fecal sample with the lactoferrin concentration of the second fecal sample. The invention of independent claims 1 and 6 is directed to a method that is sensitive enough to monitor changes in lactoferrin levels at different times in the same human to determine if the person has had a change in gastrointestinal inflammation. This allows a physician to know whether an IBD flare may be imminent before the onset of symptoms or may allow a physician to know whether a treatment, such as a pharmaceutical, has been effective in decreasing gastrointestinal inflammation using a non-invasive method.

In contrast to the invention of claims 1 and 6, the Hayes reference discusses looking at symptoms of IBD, and not lactoferrin concentration, to determine if a calcitriol treated mouse exhibited reduced symptoms of disease as compared to controls. *See* Hayes Reference, col. 23, ll. 46-60. The method taught by Hayes is limited to testing fecal samples for lactoferrin from mice with chemically-induced IBD. Applicants submit that the method for determining whether lactoferrin levels in a human have been reduced due to treatment is not discussed. Rather, Hayes states "[a] patient with symptoms of IBD is administered an effective dose of calcitriol daily until symptoms of IBD are reduced." *See id.* at col. 24, ll. 4-6. Thus, in humans, Hayes discusses determining whether calcitriol has been effective by determining if symptoms of IBD are reduced, not fecal lactoferrin levels. While the Hayes reference describes that weight, fecal

and blood hemoglobin, and fecal lactoferrin of mice are plotted as a function of time, no comparison is done and only symptoms of IBD are evaluated to determine if mice exhibit reduced symptoms of disease. Symptoms of IBD in the Hayes reference are defined as "abdominal pain, diarrhea, rectal bleeding, weight loss, fever, loss of appetite, and other more serious complications, such as dehydration, anemia and malnutrition." *See* Hayes Reference, col. 3, ll. 15-25. Nowhere in the Hayes reference are symptoms defined as lactoferrin concentrations. There is no discussion of comparing a first lactoferrin concentration from a human fecal sample for a person to a second lactoferrin concentration of a human fecal sample from the same person to monitor the inflammatory bowel disease activity of the person and determine whether treatment of the inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation.

Furthermore, the Office Action dated September 12, 2008, at Page 4, points out that the Hayes reference does not specifically detect fecal lactoferrin in human patient samples. Thus, the Hayes reference fails to teach or suggest comparing the lactoferrin concentration of a first sample with the lactoferrin concentration of a second sample from the same human to determine if the person has had a decrease or increase in gastrointestinal inflammation. The Hayes reference makes no comparison of *lactoferrin results* taken at different times from the same individual (or mouse for that matter) to determine if there has been an increase or decrease in gastrointestinal inflammation. As such, the Hayes reference fails to describe multiple features of independent claims 1 and 6.

The Murthy reference fails to overcome the deficiencies of the Hayes reference. The Murthy reference describes a dextran sulfate model that "resembles" chronic human ulcerative colitis and human colitis-associated colon cancer. *See e.g.*, Murthy Reference, page 9, col 3.

The dextran sulfate model in a mouse is not an adequate model to be used in human diagnostics for a variety of reasons. First, according to the Murthy reference, the dextran sulfate mouse model "resembles" chronic human ulcerative colitis which is limited to the large bowel. The invention of independent claims 1 and 6 is directed to both types of IBD, ulcerative colitis and Crohn's disease. Crohn's disease in humans affects both the small and large bowel. Thus, a dextran sulfate mouse model that only affects the large bowel of a mouse is not sensitive enough to be used as a diagnostic for a human disease that affects both the small and large bowel. Second, the Murthy reference teaches away from the use of the dextran sulfate mouse model in human diagnostics as "it is difficult to produce an ideal model of IBD" and "investigators must be careful in interpreting the results" of the model. *See id.* Clearly, based on the limitations of the dextran sulfate induced mouse model, the mouse model described in the Murthy reference is not sensitive enough for human diagnostics as the mouse model does not even cover the same portions of the digestive tract and there are vast anatomical differences between mice and humans.

Additionally, it would not be obvious to compare the lactoferrin concentration of a first sample with the lactoferrin concentration of a second sample from the same human over the Hayes reference in view of the Murthy reference due to the differences between human and mouse feces. Laboratory mice have a consistent diet and other parameters are controlled by researchers. On the other hand, human feces over time varies in consistency and makeup depending on a person's diet and health. It would not have been obvious in view of the Hayes reference to test the same human for a marker at different times and expect that the concentrations of lactoferrin would allow a determination of a decrease or increase in gastrointestinal inflammation.

The Uchida reference is also deficient in that it does not teach or suggest comparing the lactoferrin concentration of a first sample with the lactoferrin concentration of a second sample from the same human to determine if there has been an increase or decrease in gastrointestinal inflammation. The Uchida reference is directed to a method for diagnosing gastrointestinal tract disorders, particularly colorectal cancer, by measurement of the level of lactoferrin "in feces by immunoassay and by measurement of the level of whole-sized lactoferrin by immunoassay utilizing monoclonal antibody." Uchida reference at col. 1, lines 10–19. The Uchida reference does not compare the lactoferrin concentration of a first fecal sample with the lactoferrin concentration of a second fecal sample for the same person to determine whether the person with inflammatory bowel disease has had an increase or decrease in gastrointestinal inflammation.

Moreover, the Aguila La O reference does not overcome the above-described deficiencies of the cited references. Aguila La O is directed to studying various lactoferrin preparations to allow its use in basic studies, including the diagnosis of gastrointestinal inflammation. *See* Aguila La O Reference, Abstract. However, the Aguila La O reference does not teach or suggest comparing the lactoferrin concentration of a first fecal sample with the lactoferrin concentration of a second fecal sample from the same individual to determine if the person has had a decrease or increase in gastrointestinal inflammation.

As such, it is respectfully submitted that the Hayes, Murthy, Uchida, and Aguila La O references fail to teach or suggest each limitation set forth in independent claims 1 and 6, and, as such, claims 1 and 6 are patentable over the Hayes, Murthy, Uchida, and Aguila La O references. Claim 2 depends directly from independent claim 1, and, as such, the arguments set forth above with respect to independent claim 1 are equally applicable to this dependent claim. For at least

the reasons stated above, Appellants respectfully request that the Examiner's rejection of claims 1, 2 and 6 be reversed and the claims allowed.

B) The rejection of claims 1, 2, and 6 under 35 U.S.C. § 103(a) as being unpatentable over the Hayes reference in view of Murthy and in further view of Sugi and Aguila La O should be reversed because the Examiner has failed to establish a prima facie case of obviousness.

As discussed above, the Hayes, Murthy, and Aguila La O references fail to teach or suggest comparing the lactoferrin concentration of a first sample with the lactoferrin concentration of a second sample from the same human to determine if the person has had a decrease in gastrointestinal inflammation.

The Sugi reference discloses a method for utilizing fecal lactoferrin as a marker for disease activity in a person having inflammatory bowel disease wherein multiple readings of lactoferrin levels (at 0, 12, 24, 48, 72, and 96 hours after storage at various temperatures) are taken in a single specimen over time as an assessment of protein stability. *See*, *Sugi reference*, p. 928, col. 2 and FIG. 2. The Sugi reference, however, does not describe, either expressly or inherently, a method for monitoring a patient having inflammatory bowel disease which includes obtaining a first fecal sample from a patient at a first time and obtaining a second fecal sample after treatment of the patient's inflammatory bowel disease from the same patient at a second time later than the first time, determining the concentration of the first sample and the second sample and comparing the first lactoferrin concentration to the second lactoferrin concentration to monitor the inflammatory bowel disease activity of the patient and determine whether treatment of the inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation as recited in the method of independent claims 1 and 6. Rather, the Sugi reference describes a method wherein a first reading of a fecal sample is taken at a first time and a second reading of the same fecal sample is taken at a second time. As stated in the Office

Action, the Sugi reference "measure[s] samples at different times but does not teach multiple sample collections at different times." *See* Office Action dated 9/12/2008, Page 9. The samples obtained in the Sugi reference are viewed independently and are not compared to each other to determine whether treatment of the inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation. The Sugi reference clearly states "[t]hirteen of 41 UC patients and 16 of 34 CD patients were hospitalized two or more times, and **each admission was treated as an independent clinical course.**" *See* Sugi Reference, page 928 last line of column 1. The Sugi reference lacks any description, express or inherent, of comparing the lactoferrin concentration of a first and second sample taken from the same patient at different times in order to monitor the inflammatory bowel disease activity of the patient and determine whether treatment of the inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation.

Therefore, the Hayes, Murthy, Sugi, and Aguila La O references, whether taken alone or in combination, fail to teach or suggest the recited features of independent claims 1 and 6 discussed above. Consequently, Applicants respectfully submit that independent claims 1 and 6 are nonobvious over the Hayes, Murthy, Sugi, and Aguila La O references. Claim 2 depends directly from independent claim 1. "If an independent claim is nonobvious under 35 U.S.C. 103, then any claim depending therefrom is nonobvious." MPEP § 2143.03. Accordingly, claim 2 is nonobvious over the Hayes, Murthy, Sugi, and Aguila La O references at least by reason of its dependency from independent claim 1. For at least these reasons, it is respectfully submitted that claims 1, 2, and 6 are allowable over the cited art of record.

C) Conclusion

For at least the reasons listed above, independent claims 1 and 6 are believed to be in condition for allowance, as is claim 2 at least by virtue of its dependence upon allowable independent claim 1.

Respectfully submitted,

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CLAIMS APPENDIX

1. (Currently amended) A method for monitoring a person having inflammatory bowel disease for gastrointestinal inflammation, comprising:

obtaining a first human fecal sample from a person;

diluting said first fecal sample;

contacting said first sample with immobilized polyclonal antibodies to endogenous lactoferrin to create a first treated sample;

contacting said first treated sample with enzyme-linked polyclonal antibodies to create a first enzyme-linked antibody bound sample;

adding a substrate to the first enzyme-linked antibody bound sample to create a first readable sample;

determining the optical density of said first readable sample at 450nm;

generating a purified lactoferrin standard curve and determining a linear portion of the standard curve;

comparing said optical density of said first readable sample to said standard curve to determine a concentration of the first diluted sample; and determining whether the concentration of the first diluted sample is within the linear portion of the standard curve, wherein if the first diluted sample is within the linear portion of the standard curve, determining the concentration of total endogenous lactoferrin in said first fecal sample;

obtaining a second human fecal sample from the person at a time after the first sample was obtained;

diluting said second fecal sample;

contacting said second sample with immobilized polyclonal antibodies to endogenous lactoferrin to create a second treated sample;

contacting said second treated sample with enzyme-linked polyclonal antibodies to create a second enzyme-linked antibody bound sample;

adding a substrate to the second enzyme-linked antibody bound sample to create a second readable sample;

determining the optical density of said second readable sample at 450nm;

comparing said optical density of said second readable sample to said standard curve to determine a concentration of the second diluted sample; and determining whether the concentration of the second diluted sample is within the linear portion of the standard curve, wherein if the second diluted sample is within the linear portion of the standard curve, determining the concentration of total endogenous lactoferrin in said second fecal sample; and

comparing said lactoferrin concentration of the first fecal sample to the lactoferrin concentration of the second sample for the person to monitor the inflammatory bowel disease activity of the person and determine if the person has had a decrease or increase in gastrointestinal inflammation.

2. The assay as recited in claim 1, wherein said step of diluting said fecal sample comprises diluting said first and second samples by serial ten-fold dilutions until a measured result indicates a concentration of fecal lactoferrin that provides an optical density reading at 450 nm that is within a linear portion of the standard curve.

3-5. Canceled.

6. A method for monitoring a human having inflammatory bowel disease for gastrointestinal inflammation, the method comprising:

obtaining a first fecal sample from a human having inflammatory bowel disease at a first time;

determining the concentration of endogenous lactoferrin in said first fecal sample to obtain a first lactoferrin concentration;

obtaining a second fecal sample from the human having inflammatory bowel disease at a second time after treatment of the human's inflammatory bowel disease later than said first time;

determining the concentration of endogenous lactoferrin in said second sample to obtain a second lactoferrin concentration; and

comparing said first lactoferrin concentration to said second lactoferrin concentration to determine whether treatment of the inflammatory bowel disease has been effective in decreasing or eliminating gastrointestinal inflammation.

EVIDENCE APPENDIX

Pursuant to 37 C.F.R. § 41.37(c)(1)(ix), submitted herewith are copies of any evidence submitted pursuant to 37 C.F.R. §§ 1.130, 1.131, or 1.132 or any other evidence entered by the Examiner and relied upon by Appellants in the appeal.

NONE

RELATED PROCEEDINGS APPENDIX

Pursuant to 37 C.F.R. § 41.37(c)(1)(x), submitted herewith are copies of decisions rendered by a court or the Board in any proceeding identified in Section II pursuant to 37 C.F.R. § 41.37(c)(1)(ii).

NONE

Respectfully submitted,

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